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BPCO e patologia cardiaca

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Systemic Effects of Chronic Obstructive Pulmonary Disease

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Chronic obstructive pulmonary disease (COPD) affects various structural and functional domains in the lungs. It also has significant extrapulmonary effects, the so-called systemic effects of COPD.
Weight loss, nutritional abnormalities, and skeletal muscle dysfunction are well-recognized systemic effects of COPD. Other less wellknown but potentially important systemic effects include an increased risk of cardiovascular disease and several neurologic and skeletal defects. The mechanisms underlying these systemic effects are unclear, but they are probably interrelated and multifactorial, including inactivity, systemic inflammation, tissue hypoxia and oxidative stress among others. These systemic effects add to the respiratory morbidity produced by the underlying pulmonary disease and should be considered in the clinical assessment as well as the treatment of affected patients.

Keywords: extrapulmonary effects; multicomponent disease; oxidative stress; tissue hypoxia

Systemic Effects of Chronic Obstructive Pulmonary Disease

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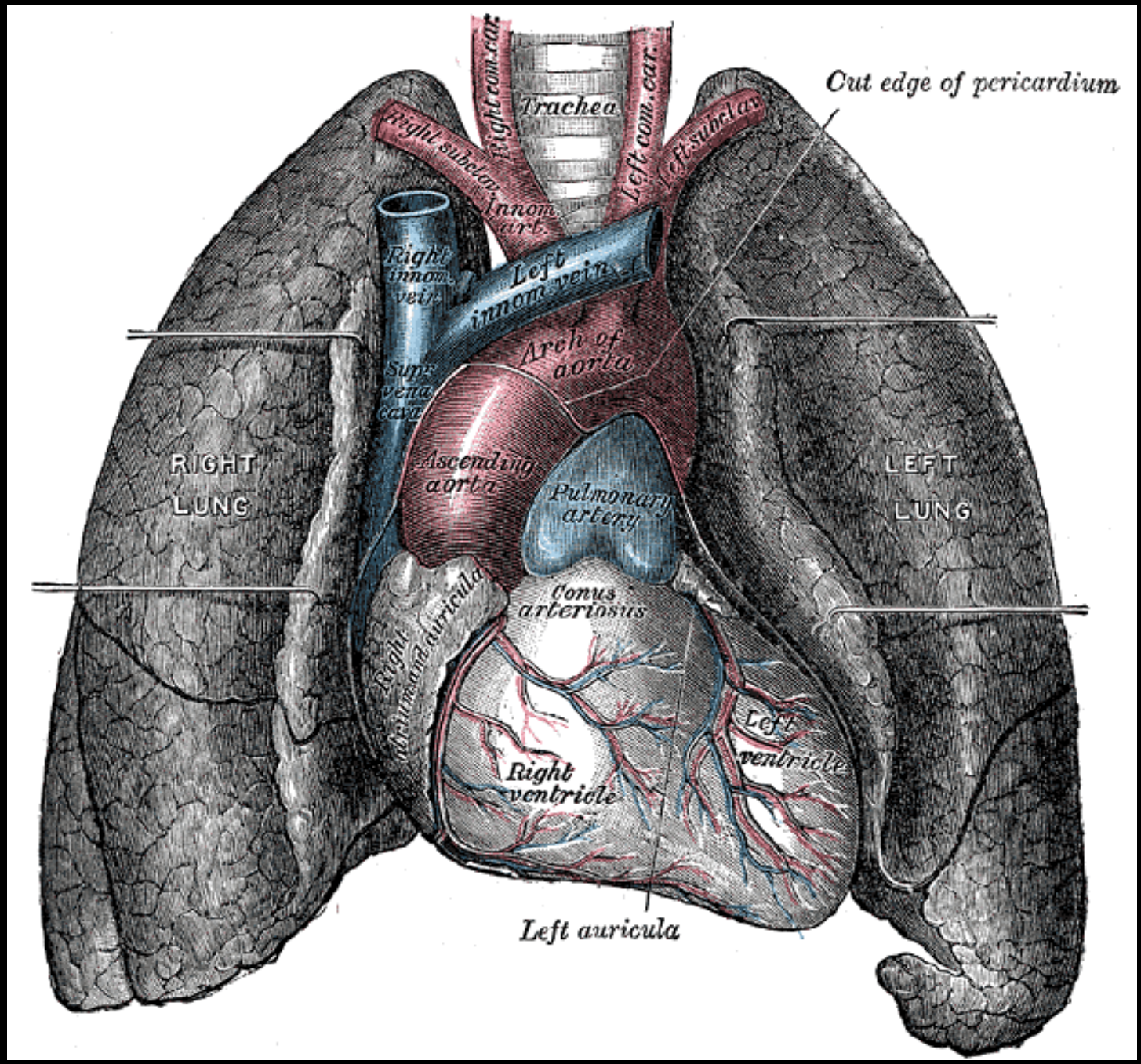
TABLE 2. POTENTIAL MECHANISMS OF THE SYSTEMIC EFFECTS DESCRIBED IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Related to COPD itself
- Spillover of pulmonary inflammation/activation of inflammatory cells in the lungs
- Tissue hypoxia (reoxygenation? pH? Pa_{CO_2}?)
- Sedentarism/inactivity due to dyspnea on exertion

Related to the cause(s) of COPD
- Smoking
- Genetic characteristics of the host

Other, as yet unidentified


Cut edge of pericardium
Right com. car.
Trachea
Left com. car.
Right subclav.
Innom. art.
Left subclav.
Right innom. vein
Left innom. vein
Arch of aorta
Sup. vena cava
RIGHT LUNG
Ascending aorta
Pulmonary artery
LEFT LUNG
Conus arteriosus
Right auricula
Left ventricle
Right ventricle
Left auricula

Effects of COPD on Cardiac Function

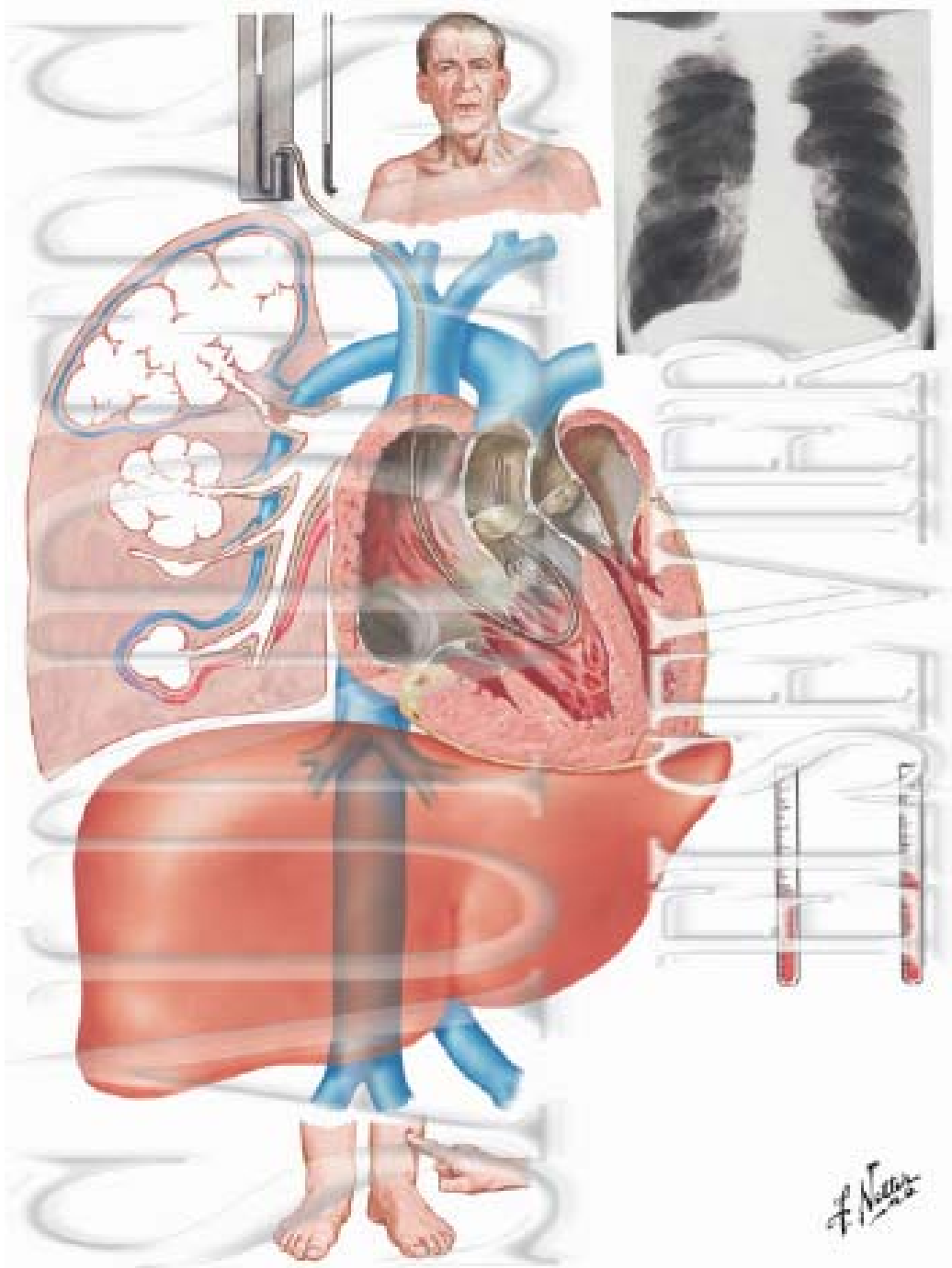
Rest		Exercise	Cardiac consequence
↑	Lung hyperinflation	↑↑↑↑	
↑	Work of breathing	↑↑↑↑	Need to deliver increased cardiac output
Normal / ↑	Intrathoracic pressure	↑↑↑↑	Decreased venous return (cardiac output)
Normal / ↑	Pulmonary hypertension	↑↑↑↑	Increased cardiac strain; limited increase cardiac output

Cuore polmonare

Caratterizzato da ipertrofia, dilatazione, ed infine insufficienza del ventricolo destro che si sviluppa come risultato dell'ipertensione cronica polmonare.

L'utilizzo più diffuso dell'ossigeno-terapia a lungo termine nei pazienti con BPCO severa e secondaria insufficienza respiratoria ipossiemica ha migliorato la sopravvivenza e probabilmente ha significativamente ridotto la prevalenza di cuore polmonare.

Nei pazienti affetti da BPCO e cuore polmare cronico si verifica un'alterazione dei fluidi e degli elettroliti determinato da un'anormale produzione di ormone natriuretico stimolato dall'ipossiemia e dalla distensione dei vasi polmonari e del ventricolo dx. Un'alterazione dell'equilibrio idro-elettrolitico si può riscontrare nei paziente affetti da BPCO anche iun assenza di ipertensione polmonare (verosimilmente per alterazioni sistemiche non ancora completamente chiarite).



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Prognosi del cuore polmonare secondario a BPCO

- 30% sopravvive a 5 anni; in media 3 anni dalla diagnosi
- Resistenze vascolari polmonari >550 dynes-sec/cm raramente sopravvive più di 3 anni
- E’ consizionato dal grado di severità della BPCO sottostante

Monaldi Arch Chest Dis
2001; 56: 6, 481–485

ORIGINAL ARTICLE

One-year mortality in elderly stable patients with COPD

P. Ranieri, R. Rozzini, S. Franzoni, M. Trabucchi, E. Clini*

ABSTRACT: *One-year mortality in elderly stable patients with COPD. R. Ranieri, R. Rozzini, S. Franzoni, M. Trabucchi, E. Clini.*

A retrospective study was performed to evaluate the risks of one-year mortality in very old hospitalized patients including those suffering from chronic obstructive pulmonary disease (COPD).

Six hundred and fifty-eight disabled patients (M=194, mean age 79.2±7.4 years) consecutively admitted to and discharged from a Geriatric Evaluation and Rehabilitation Unit (GERU) after a comprehensive rehabilitation program were studied and divided into two groups: COPD (n=337, 51%) and non-COPD (n=321, 49%). Multidimensional evaluation including information on demographics, cognitive status [Mini Mental State Examination (MMSE)], physical health [number of diseases, Greenfield's Individual Disease Severity (IDS), and number of drugs used], functional disability [Basic Activity of Daily Living (BADL), Tinetti scale, and Physical Performance Test (PPT)], and nutritional status [Prognostic Nutritional Index (PNI)] were assessed at admission. Survival rate was assessed over a 1-year period following discharge.

COPD patients mainly differed from non-COPD in terms of older age, smoking habit, number of associated diseases and drugs used. Aggregating the IDS 2-3-4 COPD classes (symptoms+functional impairment), the risk of one-year mortality was double that of the IDS 1 COPD class (symptoms only) and of non-COPD subjects (IDS 0 class) after adjusting for age, sex, disability, malnutrition, and comorbility. Moreover, IDS 2-3-4 COPD patients suffering from cor pulmonale (CP) had a fourfold 1-year risk of mortality in comparison with the IDS 1 COPD group after adjusting for the same covariates.

Hospitalized stable very old COPD patients presenting functional impairment have a higher 1-year risk of mortality than only symptomatic COPD or non-COPD subjects. The presence of cor pulmonale with COPD further increases this risk.

Monaldi Arch Chest Dis 2001; 56: 6, 481–485.

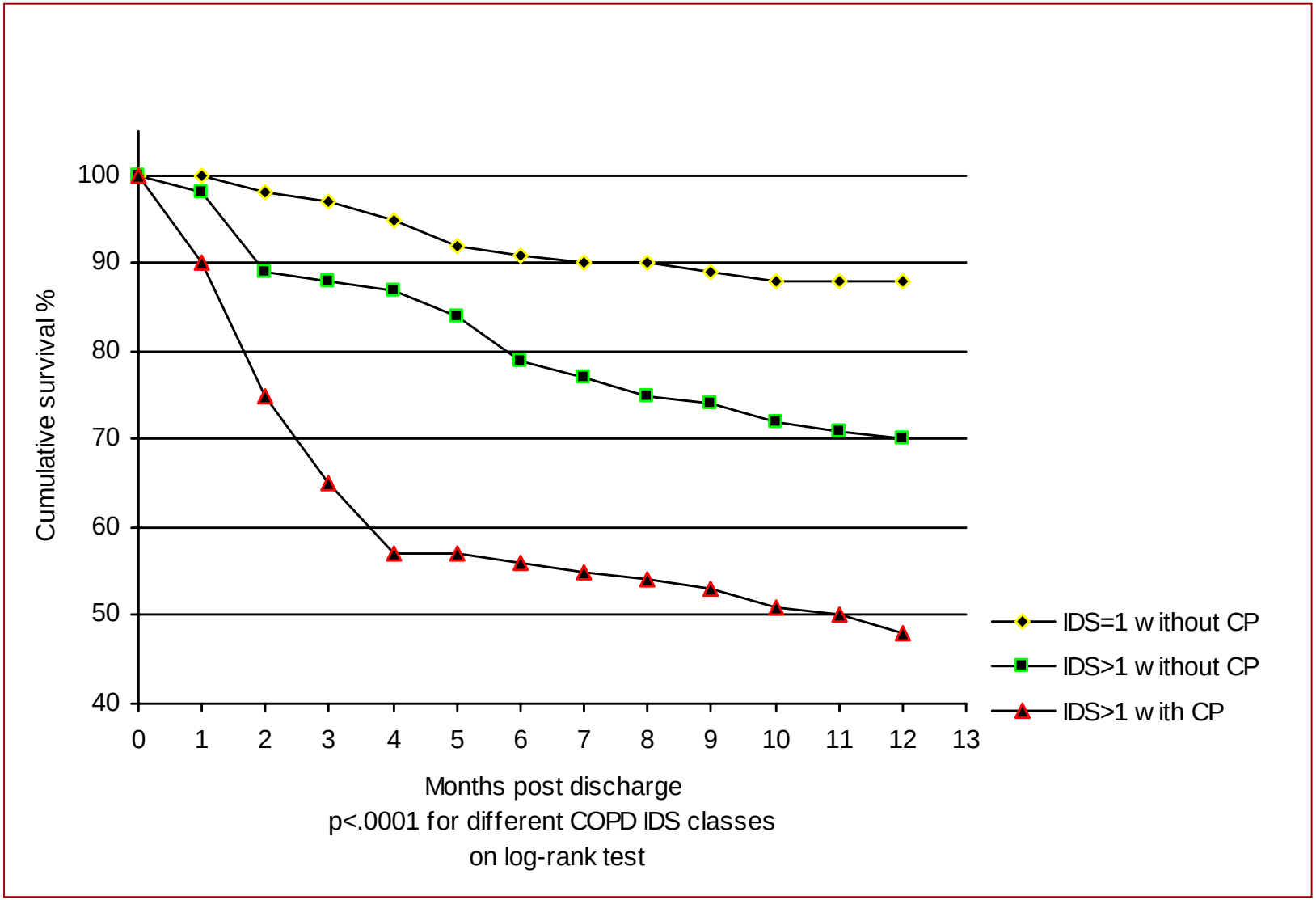


Fig. 1. – Survival across COPD-related IDS classes (IDS 1 and IDS>1) in 337 patients according to the presence of cor pulmonale (CP).

Ranieri P et al., Monaldi Arch Chest Dis 2001; 56: 6, 481–485.

In un paziente affetto da BPCO non riacutizzata, spesso clinicamente è difficile distinguere se la dispnea deriva dalla malattia cardiaca o da quella polmonare. A volte è necessario effettuare test da sforzo funzionali in grado di determinare le basi fisiologiche di origine della dispnea.

Nella pratica quotidiana spesso è anche molto difficile distinguere clinicamente la BPCO riacutizzata dallo scompenso cardiaco.

La valutazione del peptide natriuretico (brain natriuretic peptide) può risultare utile a questo scopo.

Brain Natriuretic Peptide (BNP)

- A hormone released from myocardial cells
- Both atria and both ventricles
- Inhibits weakly
  - Renin-angiotensin system (Angiotensin II)
  - Endothelin secretion
  - Systemic and renal sympathetic activity
  - Plasma aldosterone production

Brain Natriuretic Peptide (BNP)

- Higher in
  - Older >younger
  - Women > men
  - Normal weight > obese
  - Renal failure
  - Congestive heart failure (right and/or left)
- Patient is his own reference point
  - Baseline
  - Post treatment

Prognosis and BNP

- HF pts.- Highest quartile at baseline had higher mortality over 2 years at baseline (32.4 vs 9.7%) than lowest quartile.
- Following optimal medical treatment mortality increased proportionately to the level of the BNP elevation.

COPD and cardiovascular disease

Among patients with COPD, comorbidities are extremely common for a number of reasons.

COPD is a disease that increases in importance with age. Because most chronic disorders of adults also increase with age, statistically, comorbidities will be relatively common among patients with severe COPD.

The major risk factor for the development of COPD is cigarette smoking. Smoking is also a major risk factor for a large number of other illnesses, including cardiovascular disease. As a result of sharing common risk factors, patients with COPD are at further increased risk to suffer these comorbidities.

TABLE 1. DISEASES ASSOCIATED WITH CIGARETTE SMOKING

- Cardiovascular
  - **Atherosclerotic vascular disease**
    - **Coronary artery disease**
    - **Carotid vascular disease**
    - **Abdominal aortic aneurysm**
    - Mesenteric, renal, iliac
  - Peripheral vascular disease
    - Thromboangiitis obliterans (Berger's)
    - Deep venous thrombosis
    - Pulmonary embolus
  - Cardiac disease
    - Angina pectoris
    - Coronary artery spasm
    - Arrythmia
- Malignancy
  - Respiratory tract
    - **Lung cancer**
      - Squamous cell
      - Adenocarcinoma
      - Large cell
      - Small cell
    - **Laryngeal cancer**
    - **Oral cavity and pharyngeal cancer**
  - Other tissues
    - **Esophagus**
    - **Pancreas**
    - **Bladder**
    - **Uterine cervix**
    - **Endometrial**
    - Breast
    - Kidney
    - Anus
    - Penis
    - **Stomach**
    - Colorectal
    - Liver
    - **Leukemia (acute myeloid leukaemia)**
- Lung disease
  - **Chronic obstructive pulmonary disease**
    - **Emphysema**
    - **Chronic bronchitis**
  - **Asthma**
  - Other lung diseases
    - Idiopathic pulmonary fibrosis
    - Histiocytosis X
    - Respiratory bronchiolitis
    - Goodpasture's syndrome
    - Sleep apnea
    - Pneumothorax
    - **Pneumonia**
- Gastrointestinal disease
  - Peptic ulcer disease
    - **Associated with *Helicobacter pylori***
  - Gastroesophageal reflux
  - Chronic pancreatitis
  - Crohn's disease
  - Colonic adenomas
- Dermatologic disease
  - Skin wrinkling
  - Psoriasis
- Reproductive disease
  - Ovarian failure
  - **Reduced fertility (women)**
  - Pregnancy-related
    - **Preeclampsia (reduced risk)**
    - **Prematurity**
    - **Premature rupture of membranes**
    - **Placenta previa**
    - **Placental abruption**
    - Spontaneous abortion
  - **Decreased sperm quality**
  - Fetal effects
    - **Low birth weight**
    - **Impaired lung growth**
    - **Sudden infant death syndrome**
    - Febrile seizures
    - Reduced intelligence
    - Behavioral disorders
    - Atopic disease/asthma
- Effects on children of parental smoking
  - Asthma
  - Rhinitis
  - Otitis
  - Pneumonia
  - Increased risk to smoke
- Rheumatologic and bone disease
  - Osteoporosis
  - **Post menopausal**
  - **Hip fractures**
  - Rheumatoid arthritis
- Psychiatric
  - Depression
  - Schizophrenia
- Oral disease
  - **Periodontal disease**
  - Caries
  - Loss of taste
- Loss of olfaction
- Infectious disease
  - Tuberculosis
  - Pneumococcal infection
  - Meningococcal infection
- Endocrine disease
  - Altered hormonal secretion
  - Grave's disease
  - Antidiuresis
  - Goiter
- Renal disease
  - Glomerulonephritis
- Benign prostatic hypertrophy
- Eye disease
  - **Nuclear cataract**
  - Nuclear opacity
  - Macular degeneration
  - Grave's disease ophthalmopathy
- Erectile dysfunction

Modified from Reference 79. Items listed have been suggested to be associated with smoking. Those for which the Surgeon General's report found sufficient evidence to suggest a causal relationship are indicated in bold type (7).

COPD and cardiovascular disease

Epidemiologic studies evaluating the risk of heart disease have consistently shown an increased risk among patients with COPD. This is true even when the data are “corrected” for smoking.

There are several mechanisms that may account for this association:

- The lung disease may serve as a “marker” for the cardiac disease. That is, individuals who smoke and are susceptible to the injury caused by smoking may suffer both lung disease and heart disease.
- The lung disease could contribute to the development of cardiac disease, or vice versa.

Several lines of evidence suggest that inflammation can contribute to the development of atherosclerosis. Increases in C-reactive protein, for example, represent a major risk factor for cardiovascular disease. In this context, COPD is also an inflammatory disease and increases in C-reactive protein are present in patients with COPD

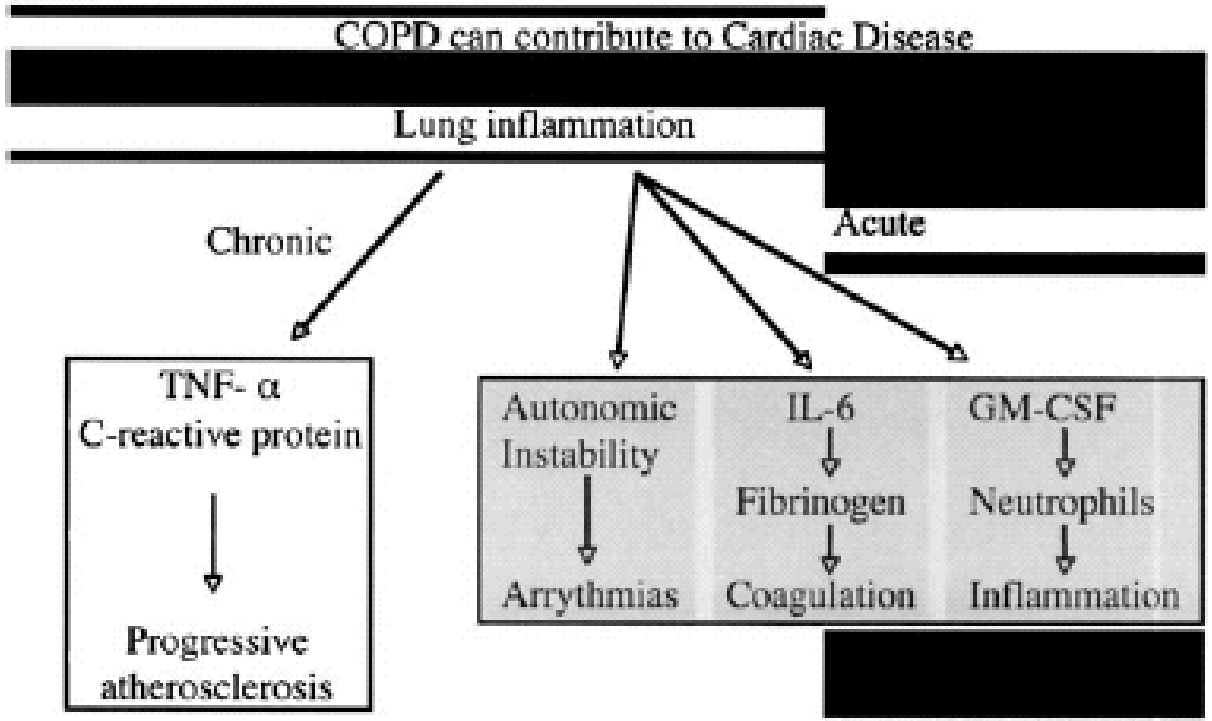
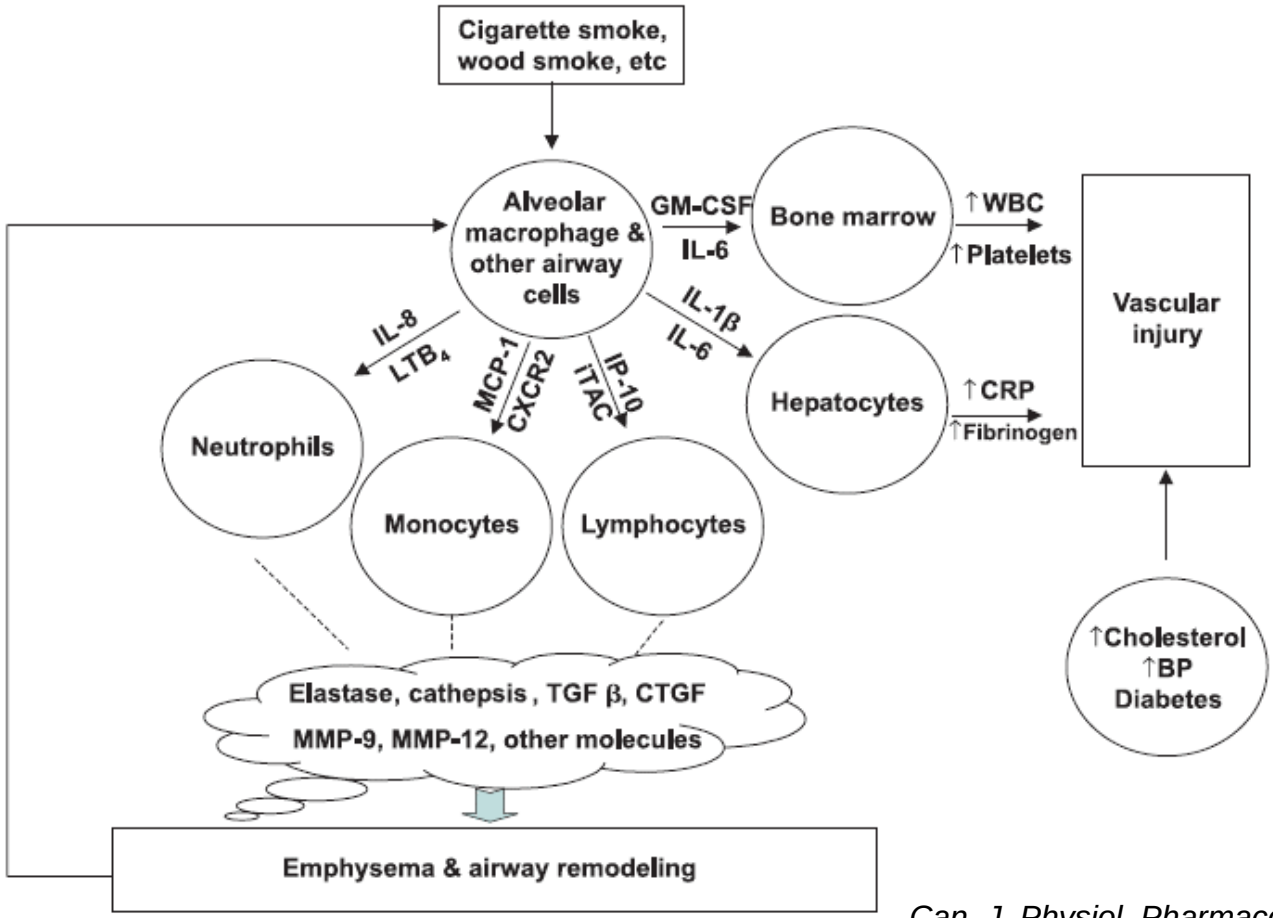


Figure 1. Chronic obstructive pulmonary disease (COPD) can contribute to cardiac disease by a variety of mechanisms. Chronic inflammation in the lung may lead to circulating cytokines and mediators (e.g. tumor necrosis factor α [TNF-α] or C-reactive protein), which may accelerate the development of atherosclerosis. Acute inflammation may predispose to the development of acute cardiac events by a number of mechanisms (*see* text for details). IL-6 = interleukin 6; GM-CSF = granulocyte-macrophage colony–stimulating factor.

Chronic obstructive pulmonary disease: a novel risk factor for cardiovascular disease[1]

Don D. Sin and S.F. Paul Man



Can. J. Physiol. Pharmacol. ***83****: 8–13 (2005)*

Chronic Obstructive Pulmonary Disease as a Risk Factor for Cardiovascular Morbidity and Mortality

Don D. Sin and S. F. Paul Man

Department of Medicine, University of British Columbia; and The James Hogg iCAPTURE Center for Cardiovascular and Pulmonary Research, St. Paul's Hospital, Vancouver, British Columbia, Canada

Chronic obstructive pulmonary disease and other disorders, associated with reduced lung function, are strong risk factors for cardiovascular events, independent of smoking. Overall, when the lowest quintile of lung function, as measured by FEV1, is compared with the highest quintile, the risk of cardiovascular mortality increases by approximately 75% in both men and women. Having symptoms of chronic bronchitis alone increases the risk of coronary deaths by 50%. Reduced ratio of FEV1 to FVC by itself is amodest independent risk factor for coronary events, increasing the risk by 30%. However, if patients have ventricular arrhythmias, the risk of coronary events is increased twofold, suggesting that the cardiotoxic effects of obstructive airways disease are amplified in those whohave underlying cardiac rhythm disturbances. In general, for every 10% decrease in FEV1, all-cause mortality increases by 14%, cardiovascular mortality increases by 28%, and nonfatal coronary event increases by almost 20%. **These data indicate that chronic obstructive pulmonary disease is a powerful, independent risk factor for cardiovascular morbidity and mortality.**

Epidemiologia BPCO

- Colpisce più del 5% della popolazione adulta con una morbilità e mortalità che è in aumento
- Colpisce più di 16 milioni di persone negli Stati Uniti e la sua prevalenza è aumentata del 41% dal 1982, mentre i tassi di mortalità aggiustati all'età sono aumentati del 17% tra il 1966 ed il 1982.
- La BPCO è responsabile di circa 750,000 ospedalizzazioni ogni anno negli Stati Uniti,e dal 10 al 20% di questi ricoveri si verifica in critical care units.
- L'OMS prevede che nel 2020 la BPCO diventerà la terza causa di morte (attualmente è la quarta) e la quinta causa di disabilità (attualmente è la dodicesima) nel mondo
- Ironicamente, i fondi di ricerca sulla BPCO del National Institutes of Health sono attualmente i più bassi tra tutti quelli impiegati per le principali cause di morte del Nord America

Sebbene questi dati epidemiologici sulla morbilità e mortalità siano allarmanti, essi rappresentano solo “la punta dell’iceberg”, dal momento che l’ostruzione bronchiale è un fattore importante che contribuisce alla morbilità ed alla mortalità per altre malattie.
Numerosi studi hanno dimostrato in molto sempre più convincente che una delle principali, ma spesso non riconosciuta, cause di morte nel paziente affetto da BPCO è la cardiopatia ischemica.

(Hansell AL et al. Eur Respir J 2003)

Larghi studi di popolazione suggeriscono che il paziente con BPCO è da 3 a 5 volte più a rischio di mortalità cardiovascolare, comprendendo circa il 50% del numero totali di morti. Sebbene spesso non riconosciuta, la riduzione della funzione polmonare si è dimostrata essere un forte predittore di mortalità cardiaca come ben più noti fattori di rischio, come ad esempio l’ipercolesterolemia.

(Hole DJ et al. BMJ 1996)

The relationship between FEV1 and cardiovascular disease

TABLE 1. BASELINE CHARACTERISTICS OF INCLUDED STUDIES REPORTED SINCE 1990 AND THE ASSOCIATION BETWEEN FEV_1 AND CARDIOVASCULAR MORTALITY

Author	Study Population	Sample Size	Age (*yr*)	Male (%)	Mean FEV_1 (*L or % of predicted*)	Current Smokers (%)	FEV_1 Categorization (*% predicted or L*)	Follow-up (*yr*)	RR of Cardiovascular Mortality (*95% CI*)	Adjusted Factors
Hole (12)	Renfrew & Paisley, UK	15,411	45–64 (range)	46	2.83*	36	Quintiles	15	1.56 (1.26, 1.92)*	Age, smoking status & history, blood pressure, serum cholesterol, BMI, social class
					1.99†		(≤ 73-75% vs. ≥ 108–113%)		1.88 (1.44, 2.47)†	
Schunemann (16)	Buffalo/Erie County, U.S.	1,195	47	46	2.8	58	Quintiles	29	2.11 (1.20,3.71)*	Age, education, smoking status, blood pressure, BMI
							(< 80% vs ≥ 109–114%)		1.96 (0.99,3.88)†	
Hospers (17)	Vlagtwedde-Vlaardingen, Netherlands	5,382	36	54	98%	55	<80% vs. ≥ 100%	~25	1.82 (1.42,2.34)	Sex, age, smoking status, BMI
Knuiman (18)	Busselton, Australia	4,277	49	49	95%*/100%†	45*/24†	10% decrease in FEV_1 percent predicted	20 to 26	1.10 (1.03, 1.18)*	Age, smoking status & history, symptoms, coronary heart disease, cardiovascular risk factors
									1.07 (1.00, 1.24)†	

* Male values.
† Female values.

In general, when the lowest quintile of FEV1 is compared with the highest quintile, the risk of cardiovascular mortality increases by approximately 75% in both men and women.

The relationship between rate of FEV1 decline and cardiovascular disease

In the Baltimore Longitudinal Study of Aging, individuals who experienced the most rapid decline in FEV1 over a 16-year follow-up were three to five times more likely to die from a cardiac cause of death than those who had the slowest decline in FEV1, after adjustments for age, baseline FEV1, smoking status, hypertension status, body mass index, and mean serum cholesterol level. Even among lifetime nonsmokers, accelerated decline in FEV1 was associated with a 5- to 10-fold increase in the risk for cardiac deaths, which suggests that the relationship between changes in FEV1 and cardiovascular events occurs independently of the effects of smoking. Whether cigarette smoking interacts and modifies the relationship between accelerated decline in FEV1 and cardiovascular outcomes, however, is unclear.

Why Are Patients With Chronic Obstructive Pulmonary Disease at Increased Risk of Cardiovascular Diseases?

The Potential Role of Systemic Inflammation in Chronic Obstructive Pulmonary Disease

Don D. Sin, MD, MPH; S.F. Paul Man, MD

Background—Chronic obstructive pulmonary disease (COPD) increases the risk of cardiovascular disease 2- to 3- fold. The factors responsible for this association remain largely unknown.

Methods and Results—We analyzed data from participants, ≥50 years of age, of the Third National Health and Nutrition Examination Survey (n=6629) to determine whether C-reactive protein (CRP) and other systemic inflammatory markers are present in participants with chronic airflow obstruction and are associated with cardiac injury. Participants with severe airflow obstruction had circulating leukocyte, platelet, and fibrinogen levels that were 460/μL (95% confidence interval [CI], 30 to 890/μL), 39 510/μL (95% CI, 21 730 to 57 290/μL), and 41.63 mg/dL (95% CI, 19.87 to 63.39 mg/dL) higher, respectively, than in those without airflow obstruction. They were also 2.18 times (95% CI, 1.46 to 3.27) more likely to have an elevated circulating CRP level. Moderate airflow obstruction was associated with smaller but still significant increases in these levels. Moderate and severe airflow obstruction was associated with increased occurrence of ischemic changes on electrocardiograms of participants. In the presence of both highly elevated CRP and moderate or severe airflow obstruction, the Cardiac Infarction Injury Score was 2.68 and 5.88 U higher, respectively, than in those without airflow obstruction and with low CRP, which suggests an additive effect of CRP and COPD on the risk of cardiac injury.

Conclusion—Low-grade systemic inflammation was present in participants with moderate to severe airflow obstruction and was associated with increased risk of cardiac injury. This may in part explain the high rates of cardiovascular complications in COPD. **(*Circulation*. 2003;107:1514-1519.)**

Key Words: chronic obstructive pulmonary disease ■ inflammation, systemic ■ C-reactive protein ■ epidemiology

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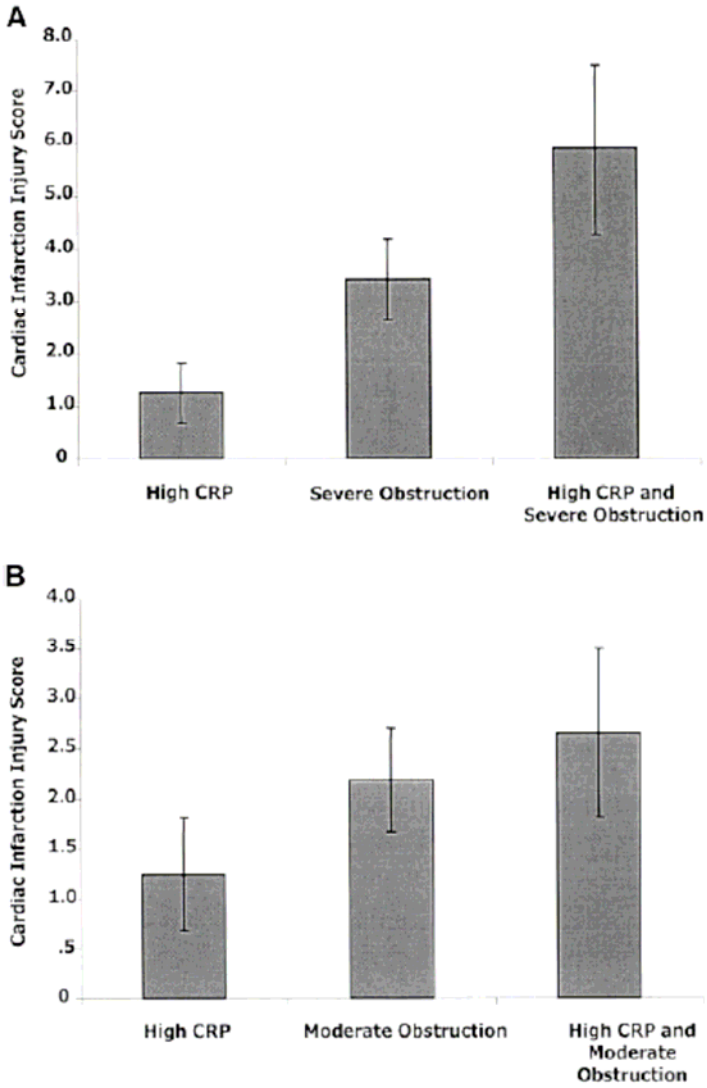
Learn and Live



A, Relationship of CRP and severe airflow obstruction to CIIS (*P* for trend=0.001). B, Interaction of CRP and moderate airflow obstruction to CIIS (*P* for trend=0.001). The shaded bars represent increases in CIIS adjusted for a variety of factors (see Methods) relative to a group without airflow obstruction and with low CRP. High CRP is defined as >1.00 mg/dL. The error bars represent SEM.

Questi dati dimostrano che la broncopneumopatia cronica ostruttiva è un fattore di rischio indipendente per malattia ischemica cardiaca, che in parte può essere spiegata attraverso l'effetto infiammatorio sistemico sull'albero vascolare coronarico.

(Circulation. 2003;107:1514-1519.)

Caratteristiche della popolazione ultra65enne dell'U.O. di Medicina stratificata in base all'assenza, alla presenza ed alla gravità di BPCO

	NO-BPCO (N=233)	BPCO (IDS 1-2) (N=135)	BPCO (IDS 3-4) (N=158)	p
	M±DS	M±DS	M±DS	
Età (anni)	*78.8±8.2*	*81.7±7.7*	*81.7±7.8*	*.000*
Durata della degenza (giorni)	*7.7±3.9*	*9.0±0.6*	*9.9±5.4*	*.000*
Autosufficienza				
Barthel Index (2 settimane prima)	81.9±28.0	80.4±29.5	74.1±31.8	.032
Barthel Index al momento del ricovero	68.4±35.4	63.1±35.9	51.0±36.3	.000
Barthel Index alla dimissione	74.3±33.3	72.8±33.6	63.2±37.0	.006
DELTA-Barthel (pre-amm)	13.5±19.9	17.3±22.9	22.7±22.7	.000
DELTA Barthel (amm/dim)	-5.0±17.1	-9.8±21.9	-12.4±21.6	.002
IADL (n. funzioni perse) (2 sett. prima)	3.1±3.1	3.0±3.0	3.7±3.1	.050
Condizioni cliniche				
Albumina sierica (g/dl)	3.7±0.6	3.5±0.6	3.4±0.5	.000
APACHE II score (0-71)	7.6±4.7	8.0±3.6	12.0±5.5	.000
Emoglobina (gr/dl)	12.1±2.1	11.9±2.0	12.6±2.0	.016
Ematocrito (%)	35.8±6.1	35.5±6.0	37.6±7.3	.007
Colesterolemia	179±56	171±48	166±50	.090
Proteina C reattiva	27.4±42	44.6±50	68.9±59	.000
Ferritina (mg/dl)	225±250	253±263	294±292	.076
Stato mentale				
Cognitività (MMSE: 0-30)	22.3±6.8	22.0±6.5	21.6±6.8	NS
Depressione (GDS: 0-15)	4.4±3.5	4.1±3.3	4.0 ± 3.0	NS

Quindi….riassumendo

- Le evidenze epidemiologiche che correlano la BPCO alla morbilità ed alla mortalità cardiovascolare sono forti e convincenti.
- Anche dopo aver aggiustato per i tradizionali fattori di rischio cardiovascolare come il colesterolo, l'ipertensione, l'obesità ed il fumo, i pazienti affetti da BPCO hanno un aumento del rischio cardiovascolare, compresa la morte, di 2-3 volte.
- Per ogni riduzione del 10% del FEV1, la mortalità cardiovascolare aumenta di circa il 28%, e gli eventi coronarici non fatali di circa il 20% nei pazienti affetti da BPCO di grado lieve-moderato.
- Anche nei pazienti con severa ostruzione bronchiale (FEV1 < 50% of predicted), la principale causa di morte rimane quella cardiovascolare.
- Il potenziale meccanismo responsabile di questa inter-relazione riguarda principalmente l'infiammazione cronica.

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doi:10.1093/eurheartj/ehi291

Clinical research

Unrecognized heart failure in elderly patients with stable chronic obstructive pulmonary disease

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Of 405 participating patients with a diagnosis of chronic obstructive pulmonary disease, 83 (20.5%, 95% CI 16.7–24.8) had previously unrecognized heart failure (42 patients systolic, 41 ‘isolated’ diastolic, and none right-sided heart failure).

In total, 244 (60.2%) patients had chronic obstructive pulmonary disease according to the GOLD criteria and 50 (20.5%, 95% CI 15.6–26.1) patients combined with unrecognized heart failure.

Conclusion Unrecognized heart failure is very common in elderly patients with stable chronic obstructive pulmonary disease. Closer co-operation among general practitioners, pulmonologists, and cardiologists is necessary to improve detection and adequate treatment of heart failure in this large patient population.

Table 1. Characteristics of 244 Elderly Patients Admitted for Non-Acidotic AECOPD.

	N	%	Mean±SD
Age (years)			81.7±7.3
Gender (males)	109	44.7	
Body Mass Index			25.1±5.5
Mini Mental State Examination (0-30)			20.7±6.8
Geriatric Depression Scale (0-15)			4.3±3.1
Instrumental Activities of Daily Living (functions lost) (0-8)			4.1±3.0
Barthel index pre-admission (0-100)			75.4±27.3
Barthel index at admission (0-100)			52.5±33.9
Barthel index at discharge (0-100)			67.2±31.3
FEV_1 (% predicted)			67.5±31.5
paO_2 (mmHg)			61.5±11.4
$paCO_2$ (mmHg)			38.5±8.9
Apache II score			9.7±4.2
Charlson index			4.0±1.9
Number of drugs at admission			5.6±2.9
Serum cholesterol (mg/dl)			172.2±43.4
Serum albumin (g/dl)			3.4±0.5
Erythrocyte sedimentation rate (ESR) (mm/h)			41.2±27.3
C-reactive protein (CRP) (mg/dl)			54.4±48.1
Hematocrit (%)			36.4± 5.5
Associated AECOPD conditions			
– None	106	43.4	
– Pneumonia	18	7.4	
– Congestive heart failure (III-IV NYHA)	82	33.6	
– Pulmonary embolism	2	0.8	
– Others	26	14,8	
Prevalence of comorbidity			
– Hypertension	139	59.9	
– Ischemic heart disease	38	15.6	
– Cerebrovascular disease	81	33.2	
– Vascular peripheral disease	57	23.4	
– Dementia	38	15.6	
– Diabetes	81	33.2	
– Malignancies	53	21.7	
Length of stays (days)			8.8±4.8
In-hospital mortality	4	2	
Transferred to Intensive Care Unit (ICU)	15	6	

Predictors of 6-Month Mortality in COPD Patients Discharged from a Medical Ward following Acute Non-Acidotic Exacerbation.

Ranieri P et al., JAGS 2007 (submitted).

Una volta riconosciuto che la malattia cardiaca e la malattia polmonare sono frequentemente concomitanti e possono essere interconnesse fisiopatologicamente, che raccomandazioni posso essere date al clinico che quotidianamente cura questi pazienti?

Sfortunatamente esiste una mancanza di specifiche raccomandazioni per il management dei pazienti con BPCO e concomitante malattia cardiaca.
Le più recenti linee guida (Global Initiative for Chronic Obstructive Lung Disease (GOLD) ed dell' American Thoracic Society/European Respiratory Society) sebbene riconoscano che la malattia cardiaca è spesso presente come comorbilità della BPCO, non forniscono specifiche e dettagliate raccomandazioni di come nella pratica clinica questi pazienti devono essere valutati e trattati.
Questo deriva principalmente dal fatto che le linee guida di per sé si basano su dati "evidence based" e nella maggior parte dei trial clinici i pazienti sono selezionati in modo da escludere quelli più complessi e con maggiore comorbilità.

Le linee guida dell' American College of Cardiology/American Heart Association per la valutazione ed il trattamento dello scompenso cardiaco nell'adulto, anch'esse considerano la frequente associaizone tra lo scompenso cardiaco e la malattia polmonare e propongono il Test da sforzo con misurazioni emogasanalitiche, possibile attraverso un cateterismo dx (!!!), come mezzo per aiutare il clinico a determinare quale delle due componenti, la cardiaca o la polmonare, è preponderante in un determinato paziente.

La BPCO non è descritta nelle linee giuda dell’OBESITA’ e dell’IPERTENSIONE e non è inclusa nella lista dei fattori di rischio cardiaco delle linee guida delle malattie cardiovascolari.

In assoluto esiste una mancanza di raccomandazioni che guidano il clinico nella care specifica dei pazienti con BPCO che sono affetti da obesità, ipercolesterolemia, ipertensione arteriosa, o altri fattori di rischio.

OBESITA’

- Sebbene l’obesità sia un chiaro fattore di rischio di mortalità per malattia cardiaca, la relazione tra peso corporeo e rischio di BPCO è molto più complessa.
- Nei pazienti affetti da BPCO la mortalità aumenta al diminuire del peso corporeo.
- La perdita di peso nei pazienti affetti da BPCO è stata attribuita all’effetto delle citochine circolanti (in particolare al Tumor-necrosis factor). In aggiunta a ciò il fabbisogno calorico di questi pazienti aumenta a causa del maggior lavoro respiratorio e muscolare. La depressione del tono dell’umore o altri fattori che compromettono la nutrizione possono inoltre giocare un ruolo importante. Come tutti questi fattori interagiscano con la malattia cardiaca e se essi sono rilevanti per i pazienti affetti da BPCO in sovrappeso, rimane tutto da determinare.
- Tuttavia anche i pazienti BPCO in sovrappeso posso avere una diminuzione della massa magra, responsabile di ridotte performance respiratorie. La semplice raccomandazione di perdere peso nei pazienti affetti da BPCO e sovrappeso, potrebbe non essere corretta.

REHABILITATION AND EXERCISE

- Both patients with COPD and patients with cardiac disease can benefit from exercise training.
- Rehabilitation programs can substantially reduce cardiac risk and should, therefore, be part of the management program of all patients who are at hazard. Rehabilitation can, moreover, dramatically improve quality of life among patients with COPD.
- How best to implement the rehabilitation program among patients with concurrent cardiac and heart disease is not fully determined. In this regard, the intensity of the exercise training is a key factor in determining the training benefit. Even modest degrees of COPD, however, can compromise training.
- Additional studies of exercise training and rehabilitation among patients with concurrent cardiac disease and COPD are needed to determine whether interventions for COPD that may be otherwise asymptomatic may improve training enefits and result in cardiac benefits.

Clinical Approach to Patients with Chronic Obstructive Pulmonary Disease and Cardiovascular Disease

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NOVEL APPROACHES TO THERAPY

There are several reasons to consider novel approaches to the development of cardiac and pulmonary diseases by evaluating them together. The physiologic interdependence of the organ systems makes it possible that treatment of otherwise mild cardiac disease may benefit respiratory function in patients with COPD and vice versa. This may result in novel endpoints in clinical trials. It is also possible that cardiac and respiratory disease can share mechanisms. For example, antiinflammatory therapy treatment of COPD may benefit cardiovascular disease, and statin therapy for heart disease could have antiinflammatory effects on lung disease. Specific information on the management of patients with concurrent cardiac and pulmonary disease would, at a minimum, be helpful to the clinician.

TABLE 2. CARDIAC RISK FACTORS TO CONSIDER IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Hypertension
Diabetes
Cholesterol
Renal compromise (glomerular filtration rate < 60 ml/minute)
Microalbuminemia
Obesity
Physical inactivity
Family history
Suggested lung assessment in patients at risk for cardiac disease
FEV_1
FVC or FEV_6
If any abnormalities present, consider
Post-bronchodilator FEV_1
Lung volumes
DL_{CO}
Exercise challenge (with and without bronchodilator*)

Definition of abbreviation: DL_{CO} = carbon monoxide diffusion coefficient.

* It is possible that reduced tachypnea will lead to increased exercise tolerance. The clinical implications of this are uncertain. *See* text for details.

Domande ancora senza una risposta……..

- Potrebbe il trattamento antiinfiammatorio polmonare ridurre il rischio di eventi cardiaci acuti?
- Potrebbe il trattamento antiinfiammatorio po
  de
- Po
  po
- Il trattamento della malattia cardiaca può influenzare la progressione della malattia polmonare?

Il geriatra nell'ambito di questo scenario ha senza dubbio competenze, sensibilità e cultura per contribuire in modo concreto e pratico alla ricerca in questo campo